بسم الله الرحمن الرحيم

Kidney Disease , BMI: Relationship

By

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Agenda

- **BMI**
- **Obesity and Kidney Disease**
  - **Physiopathological Mechanisms**
  - **Effects**
- **BMI and CKD**
- **BMI and ESRD**
  1. BMI and Hemodialysis
  2. BMI and Peritoneal dialysis
  3. BMI and renal transplantation

About BMI

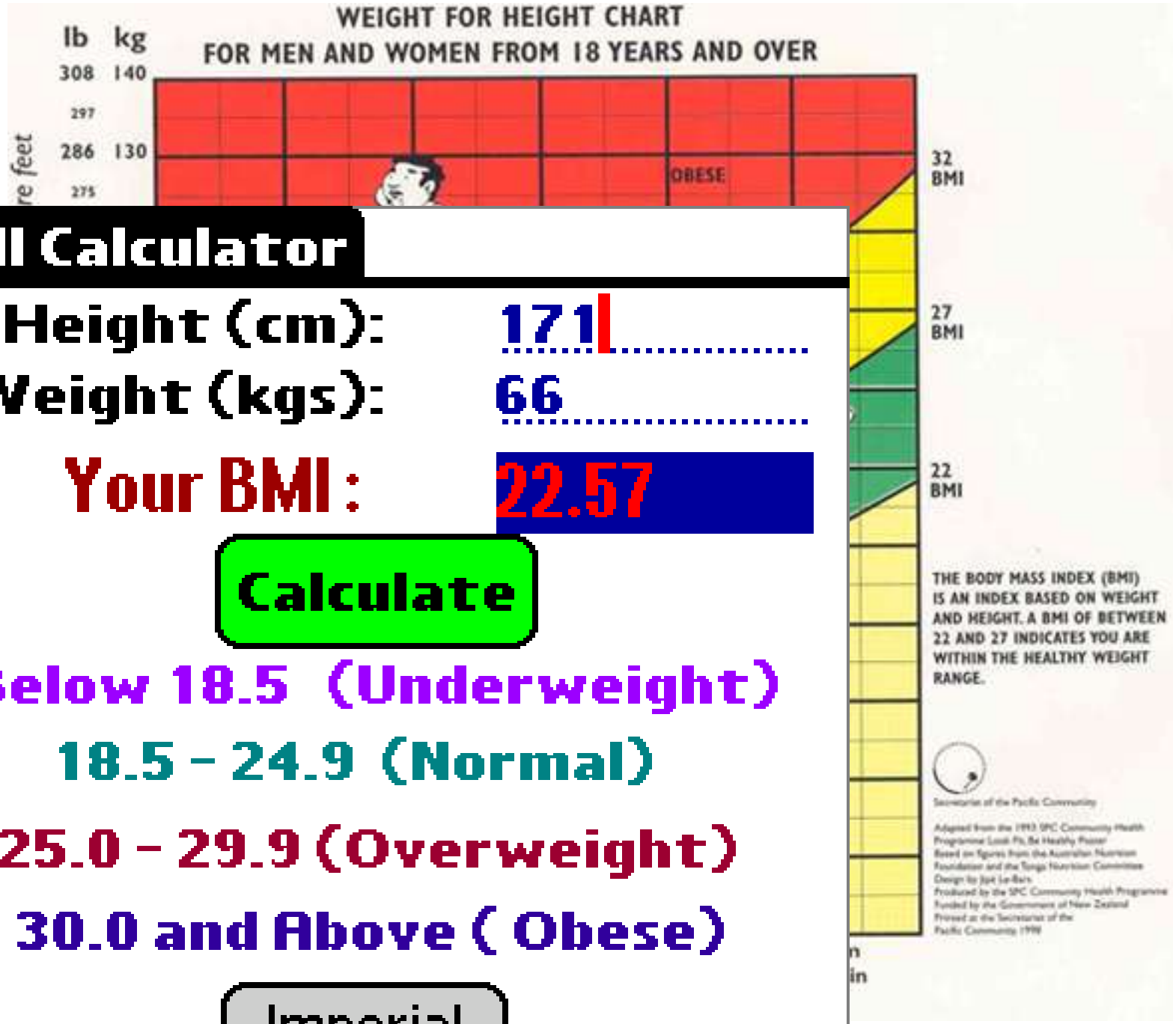
- What is meant by BMI?
- Is it of VALUE ?
- What is the History Behind It?
- It is suitable for all ages?
- Is it an ACCURATE test?
- Is it Ideal for measuring body FATNESS?
- What is the limitation of it?
- What is the sensitivity and specificity for it?

Body Mass Index (BMI)

- Statistical measurement using a person's height and weight to determine their "healthy" body weight.

- Easy to determine
- widespread in use.
- Mathematically →
- BMI chart .

$$\mathrm{BMI} = \frac{\mathrm{weight\ (kg)}}{\mathrm{height}^2\mathrm{(m^2)}}$$



BMI Calculator

Height (cm): 171

Weight (kgs): 66

Your BMI: 22.57

Calculate

Below 18.5 (Underweight)

18.5 - 24.9 (Normal)

25.0 - 29.9 (Overweight)

30.0 and Above (Obese)

Imperial

- What is meant by BMI?
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- **What is the History Behind It?**
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The History Behind It

- 1840 by **Adolphe Quetelet**.
- He was a Belgian "Renaissance Man".
- He took a person's mass in kilograms and divided it by their height in meters, squared. This gave BMI, which was originally called the Quetelet Index.

The History Behind It

- The term BMI came about in the 1970s in a paper by **Ancel Keys**.
- He was a scientist who looked at the influence of diet on health.
- He coined the categories for BMI: underweight, healthy, overweight and obese.

- What is meant by BMI?
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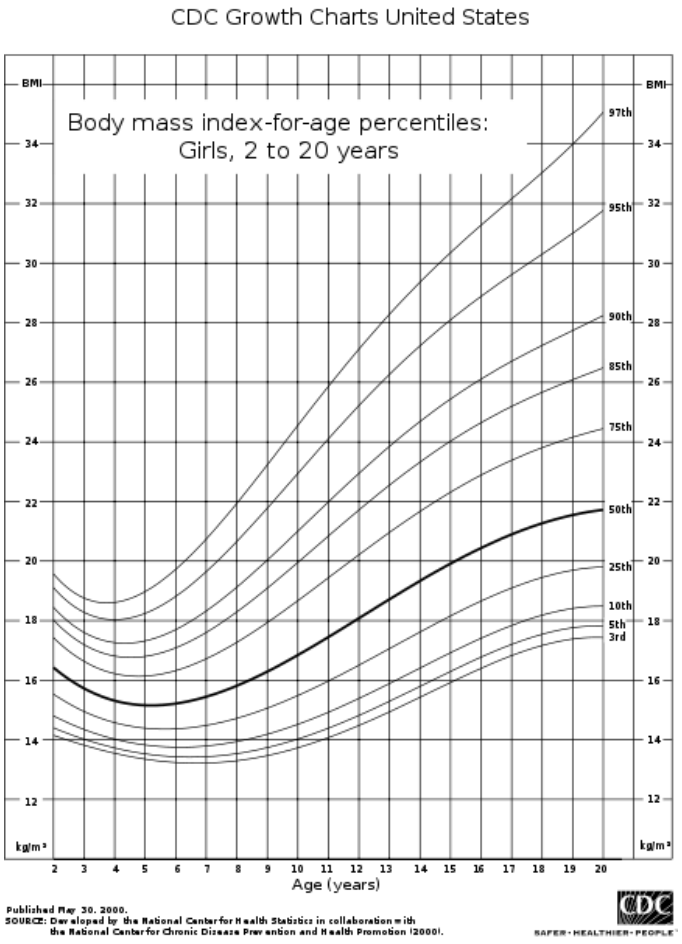
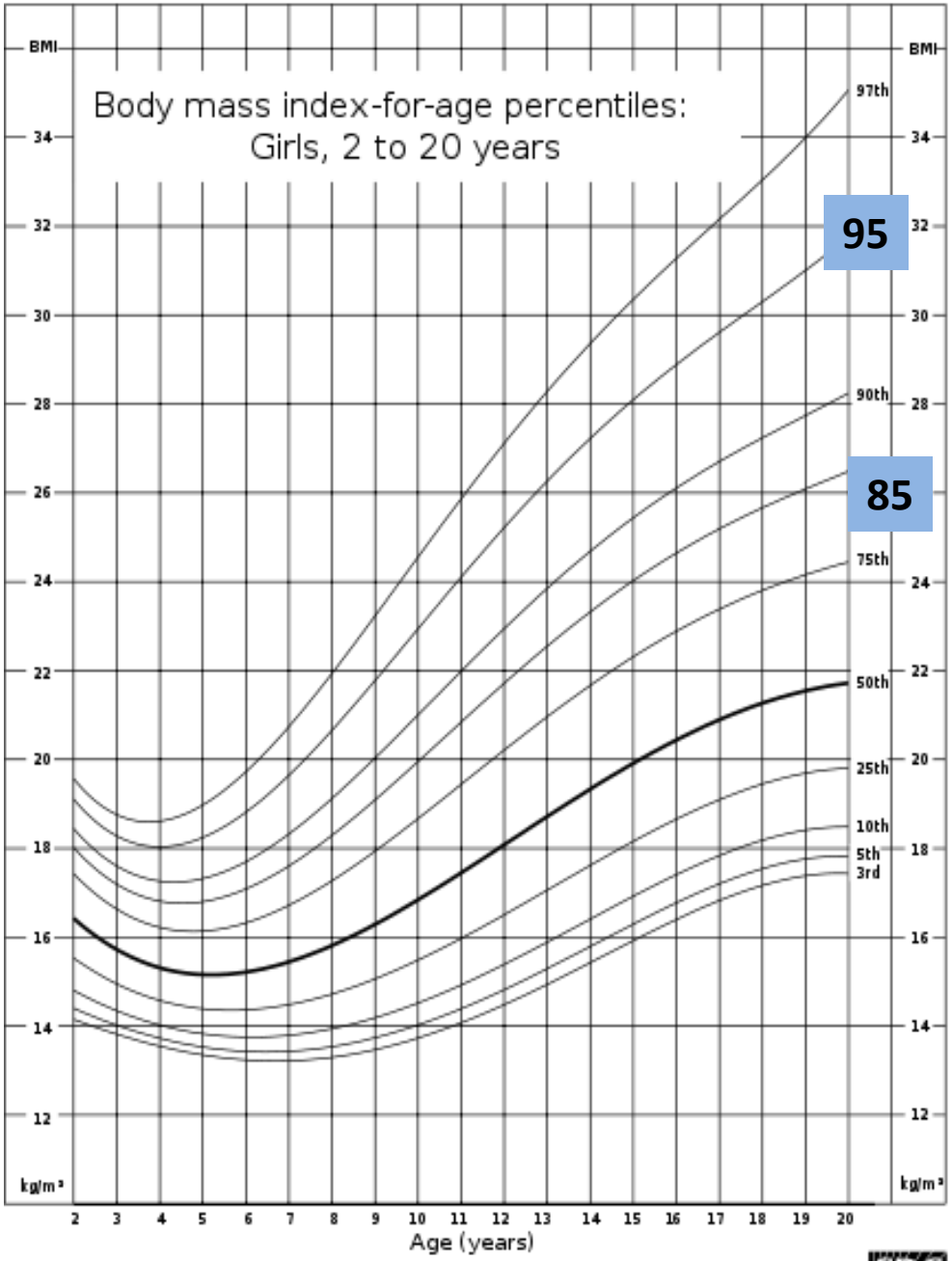
BMI for The Under 20s

- This compares the BMI total with the general averages for children and not adults.
- Calculate BMI on the BMI Index and then plot it and the age on the chart.
- Anywhere between the 5th and 85th percentile is considered “healthy”.



CDC Growth Charts United States



BMI of 30

BMI of 25

Published May 30, 2000.
SOURCE: Developed by the National Center for Health Statistics in collaboration with

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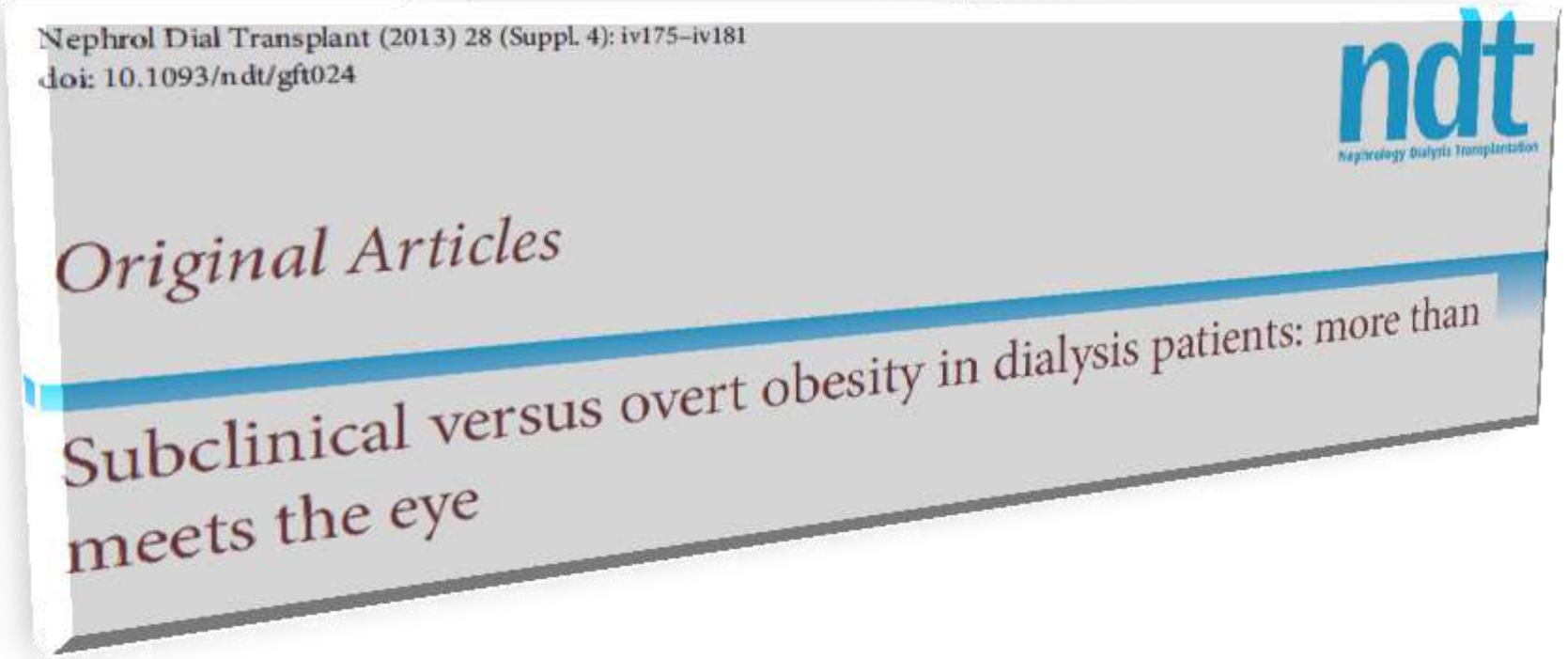
BMI is controversial

because it does not actually measure a person's percentage body fat. It just displays what a healthy weight range would be for a person's height



- A BMI of <30 kg/m2 does not exclude the presence of excess adiposity.
- Subclinical obesity is a frequent condition in dialysis patients.
- Among those with CKD; a better test to classify obesity (skinfold thickness) than (BMI)

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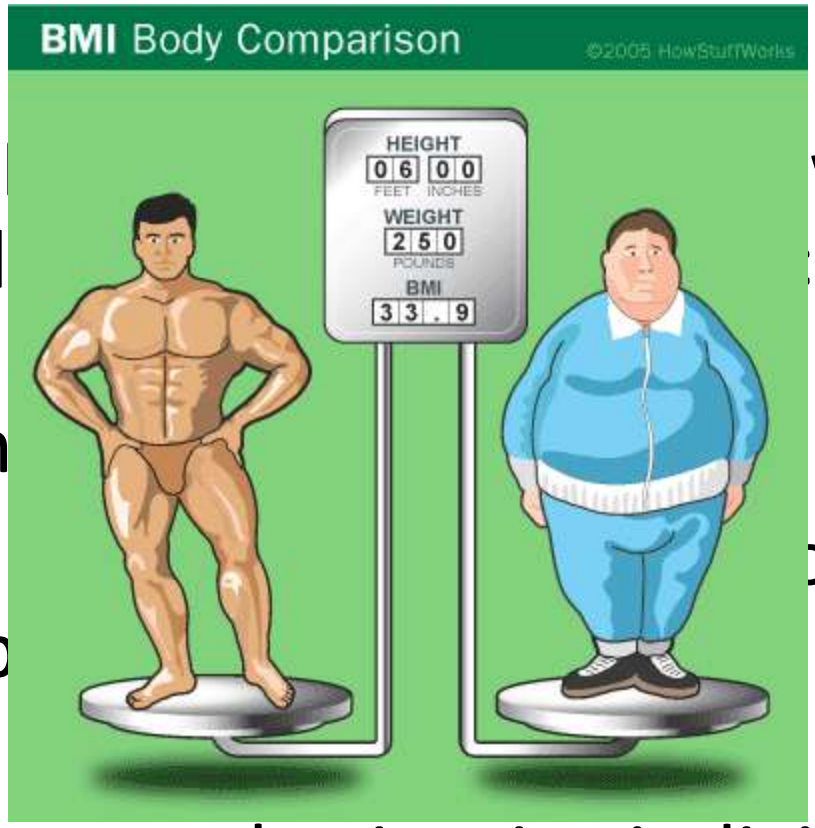
Nephrol Dial Transplant (2013) 0: 1–3
doi: 10.1093/ndt/gft266

ndt
Nephrology Dialysis Transplantation

In Focus

Diagnosis of obesity in chronic kidney disease: BMI or body fat?



Although BI with body fat, but this ind components of the body the regional distribution

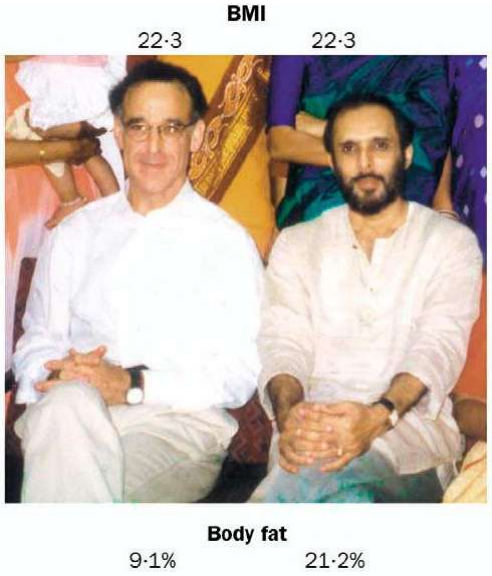
Therefore, ow sensitivity and/or sp groups of individuals,

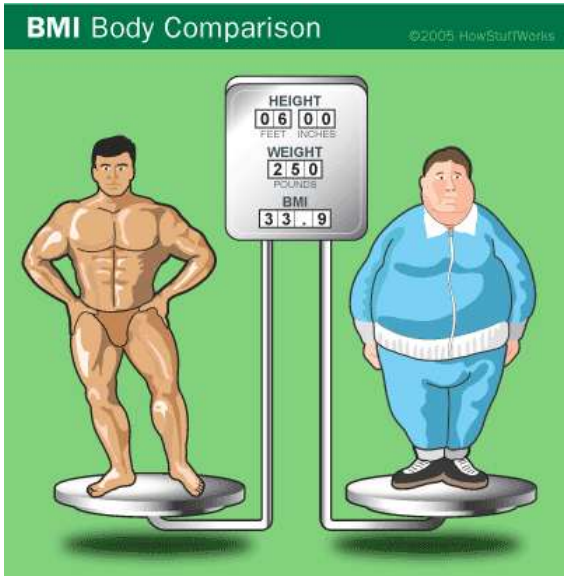
- Overestimate obesity in individuals with a high degree of muscle mass or with volume overload.(overweight BUT not overfat)
- Underestimate obesity in the elderly and in patients with wasting conditions

The consequences vary by race-ethnicity: Body fat composition in the East vs the West

(Yajnik & Yudkin 2004)

But…..these misclassified persons are uncommon relative to the population as a whole





Obesity and Kidney Disease

Obesity has rapidly become a major challenge to health care systems worldwide.

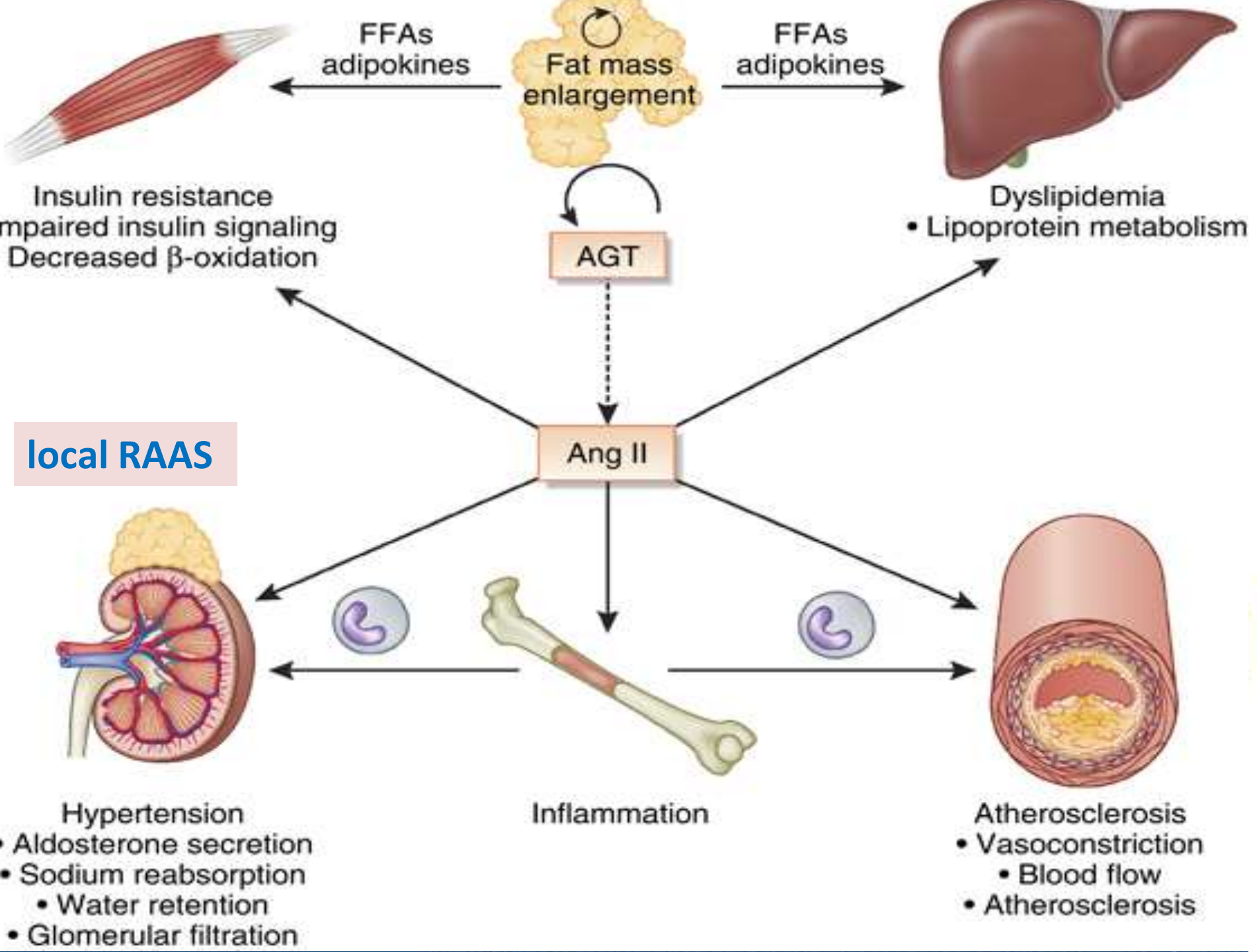
According to the WHO, more than 1.4 billion people worldwide were overweight and another 500 million were obese in 2008.

In the United States, approximately two-thirds of the adult population is overweight and nearly one-third is obese.

http://www.cdc.gov/obesity/data/prevalence-maps.html

*Prevalence estimates reflect BRFSS methodological changes started in 2011. These estimates should not be compared to prevalence estimates before 2011.[+] Guam and Puerto Rico were the only US territories with obesity data available on the 2013 BRFSS.

Physiopathological Mechanisms of Obesity Related Vascular and Renal Damage


FFAs
adipokines
Fat mass
enlargement
FFAs
adipokines
Insulin resistance
mpaired insulin signaling
Decreased β-oxidation
AGT
Dyslipidemia
• Lipoprotein metabolism
local RAAS
Ang II
Hypertension
• Aldosterone secretion
• Sodium reabsorption
• Water retention
• Glomerular filtration
Inflammation
Atherosclerosis
• Vasoconstriction
• Blood flow
• Atherosclerosis

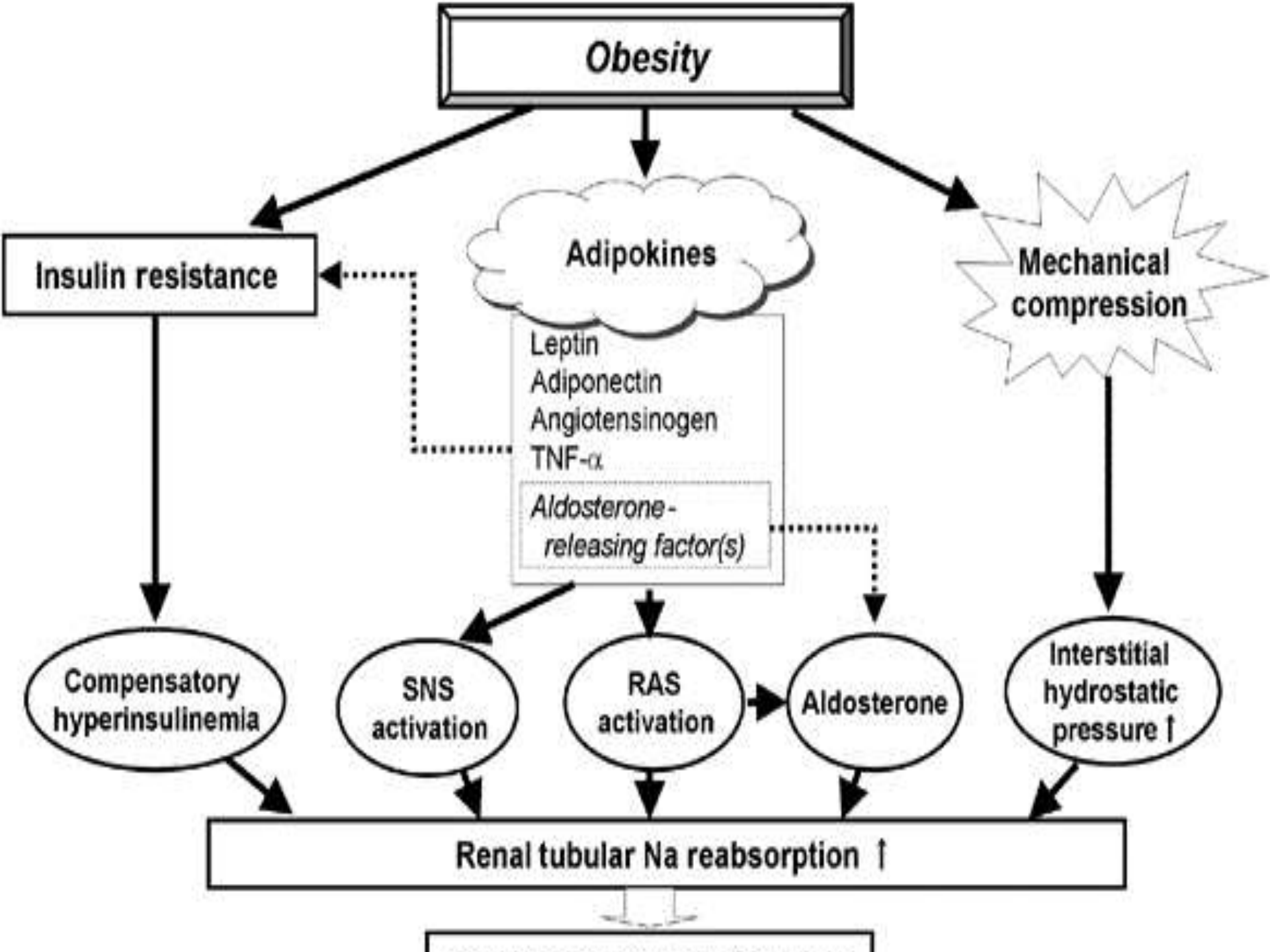
An excess of adipokines reduces the amount of glucose-transporting intracellular proteins (Glut 1-Glut 4). Concurrently, obesity increases cell endoplasmic reticulum stress. The combination of these two factors causes an increase in tissue resistance to insulin, which results in **hyperinsulinism.**

Hyperinsulinism acts on various biomolecules and their receptors causing:

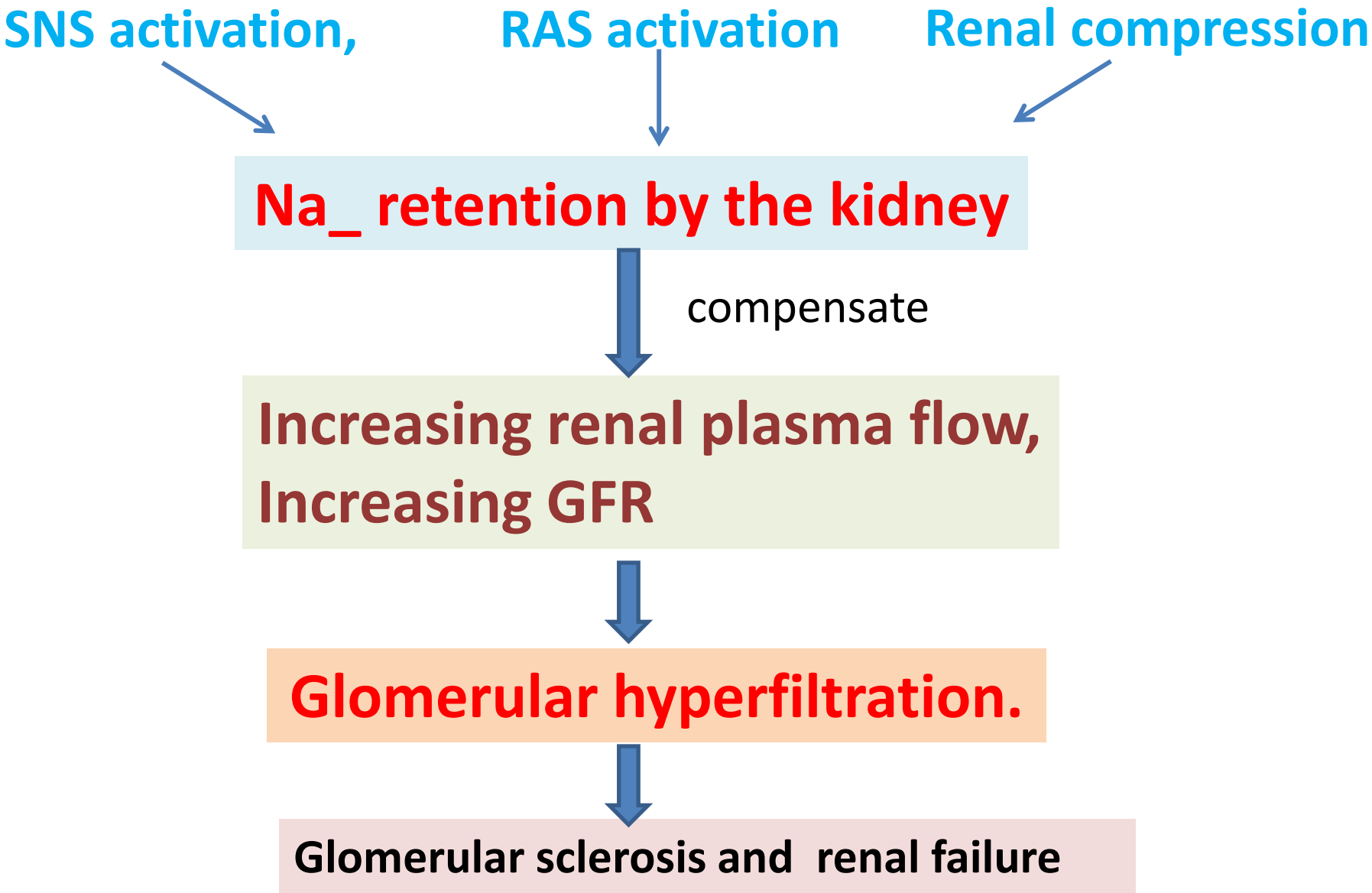
- Increased sympathetic activity, salt sensitivity and AgII and ET levels and decreased nitric oxide and natriuresis, leading to systemic hemodynamic changes, hypertension and systemic vascular damage;

- Increased glomerular capillary pressure, hyperfiltration, podocyte damage and albuminuria;

- Release of mitogenic factors (IGF-1, TGF-β, PAI-1) that induce mesangial cell proliferation and greater glomerular matrix deposition.


Obesity
Insulin resistance
Adipokines
Mechanical compression
Leptin
Adiponectin
Angiotensinogen
TNF-α
Aldosterone-releasing factor(s)
Compensatory hyperinsulinemia
SNS activation
RAS activation
Aldosterone
Interstitial hydrostatic pressure ↑
Renal tubular Na reabsorption ↑

OBESITY


SNS activation,
RAS activation
Renal compression
Na_ retention by the kidney
compensate
Increasing renal plasma flow,
Increasing GFR
Glomerular hyperfiltration.
Glomerular sclerosis and renal failure

Sleep apnea and obesity

75% of people with sleep apnea are obese. Apnea → hypoxia → "hypoxia-induced factors (HIF)"; these allow cells to adapt to low oxygen levels.

The persistence and intensity of this physiological mechanism causes inflammatory cell infiltration and higher levels of TGF-β, CTGF, metalloproteinase inhibitors and PAI-1, which cause:

inflammation, apoptosis, podocyte damage, and renal fibrosis

Effects of overweight and obesity on the kidney

- ❑Hemodynamic
- ❑Structural
- ❑Pathologic
- ❑Chronic kidney disease
- ❑End-stage renal disease
- ❑Other

Effects of overweight and obesity on the kidney

- **Hemodynamic**
  - **1- ↑Effective plasma flow**
  - **2- ↑ Glomerular filtration rate**
  - **3- ↑ Glomerular filtration fraction**
  - **4- ↑ Albuminuria**

State of renal vasodilatation involving, mainly or solely, the afferent arteriole.

- Chronic kidney disease
- End-stage renal disease
- Other

Urinary albumin excretion

In obese state, two main cellular targets associated.

①Structural changes to the glomerulus allowing more protein to enter the filtrate.

②The proximal tubules are unable to endocytose the increase in protein load .

(Thus, both the glomerulus and proximal tubules are sites of dysfunction in proteinuric nephropathy.)

This effects are magnified in the presence of hypertension and DM.

This effect occur in both non diabetic and diabetic overweight.

Effects of overweight and obesity on the kidney

- Hemodynamic
- Structural
- **Pathologic**
  - **Glomerulomegaly**
  - **Glomerulosclerosis**
  - **Obesity related glomerulopathy**
- Chronic kidney disease
- End-stage renal disease
- Other

Obesity-Related Glomerulopathy

Definition:

- ✓ presence of glomerulomegaly and/or FSGS
- ✓ in a patient with a BMI ≥ 30 kg/m2
- ✓ in the absence of other causes of FSGS such as HIV, heroin abuse, and reduced renal mass.

Obesity and Focal Segmental Glomerulosclerosis

Clinically:

- ✓ Nephrotic range proteinuria is lower,
- ✓ Serum albumin higher,
- ✓ Serum cholesterol lower,
- ✓ Edema less severe, and
- ✓ Progression to end-stage renal disease slower

Histologically:

- ✓ Larger glomerular size
- ✓ Mild foot process fusion.

BMI and CKD

Case

50yo male presents for evaluation of proteinuria

Hx : HTN Family Hx of T2DM

Ex: BP 155/94 BMI 42

Labs : BUN12, CRE 1.4, FBS 123, TG 145

UA + 4 protein

Does obesity has a role in this patient CKD?

Does obesity alter progression in CKD?

Does obesity affecting mortality in CKD?

Effects of overweight and obesity on the kidney

Hemodynamic

Structural

Pathologic

Chronic kidney disease

1. ↑ Onset of kidney disease
2. ↑ Progression to kidney failure
3. ↑ Proteinuria

End-stage renal disease

Other

Predictors of New-Onset Kidney Disease in a Community-Based Population

Caroline S. Fox, MD, MPH
Martin G. Larson, ScD
Eric P. Leip, MS
Bruce Culleton, MD
Peter W. F. Wilson, MD
Daniel Levy, MD

Context Kidney disease is associated with an increased risk for the development of cardiovascular disease and end-stage renal disease; however, risk factors for kidney disease have not been well studied.

Objective To identify predictors of the development of new-onset kidney disease.

Design, Setting, and Participants A community-based, longitudinal cohort study of 2585 participants who attended both a baseline examination in 1978-1982 and a follow-up examination in 1998-2001, and who were free of kidney disease at baseline.

Main Outcome Measures Kidney disease was assessed by the Modification of Diet in Renal Disease Study equation and defined by a glomerular filtration rate (GFR) in the fifth or lower percentile (≤59.25 mL/min per 1.73 m² in women, ≤64.25 mL/min per 1.73 m² in men). Stepwise logistic regression was used to determine the impact of risk factors on the occurrence of new-onset kidney disease. Baseline and long-term, 12-year, averaged risk factor models were explored.

Results At baseline, there were 1223 men and 1362 women, with a mean age of 43 years, who were free of preexisting kidney disease. After a mean follow-up of 18.5

HYPERTENSION AND DIABETES are the leading causes of end-stage renal disease (ESRD).[1] Among individuals who develop ESRD, the risk of cardiovascular disease is 10 to 20 times higher than the general population,[2-4] and increased risks are evident ever
disease.[5-7] There are
275 000 patients wi
United States[1] and it
an additional 8 millio
kidney disease (defin
lar filtration rate [GI
min per 1.73 m²).[8] Be
ease often progresses
attendant complicati
cation of precursors
is important, with the belief that interventions will prevent or delay the progression to ESRD.

We previously reported that age,

Cohort of 2585 patients
Followed for 20 years,
The odds ratio for new onset of CKD was 1.23 per one standard ncrease in BMI.

Conclusions Established cardiovascular disease risk factors are associated with the development of new-onset kidney disease. Patients with a mildly reduced GFR should be monitored for progression to kidney disease.

Association between body mass index and chronic kidney disease: A population-based, cross-sectional study of a Japanese community

Ikuo Nomura[1]
Johji Kato[2]
Kazuo Kitamura[1]

[1]Department of Internal Medicine, Circulatory and Body Fluid

Abstract: Chronic kidney disease (CKD) has recently been recognized as a risk factor for cardiovascular disease, while the mechanism by which CKD develops remained to be clarified. In the present study, we conducted a cross-sectional, community-based study to identify the factor(s) associated with CKD. We examined 1978 local residents of the Kiyotake area of Japan (697 males and 1281 females; age, 60.8 ± 11.0 years; mean ± SD), who had an annual health

1978 local residents of the Kiyotake area of Japan.

BMI was a significant parameter independently correlated with CKD in both genders.

Thus, increased BMI is associated with CKD independently of blood pressure, serum lipid and glucose levels in the general population.

Nephrol Dial Transplant (2008) 23: 1910–1918
doi: 10.1093/ndt/gfm878
Advance Access publication 21 December 2007

Original Article

NDT
Nephrology Dialysis Transplantation

Association between body mass index and chronic kidney disease in men and women: population-based study of Malay adults in Singapore

Anoop Shankar[1,2], Chenlei Leng[3], Kee Seng Chia[1,2], David Koh[1,2], E. Shyong Tai[2,4], Seang Mei Saw[1,2,5], Su Chi Lim[2,6] and Tien Yin Wong[5,7]

Relationship between BMI and CKD, by gender, in a population based study of Malay adults (*n* = 2783) from Singapore.

(Higher BMI levels were positively associated with CKD among men but not women).

(male gender-specific association between BMI and CKD).

Why men at higher risk of developing kidney disease?

in some animal studies, **oestrogens**

1. **Reduce mesangial proliferation and synthesis of types I and IV collagen**, in murine cell cultures.
2. Stimulate renal nitric oxide generation and to have antioxidant properties .

In contrast, **androgens** may

1. **Increase arterial pressure**, either by having a direct effect to increase proximal tubular reabsorption or by activation of the RAS .
2. BMI reflects visceral fat (a critical source of cytokines) more in males than in females .

Inverse association between body mass index and chronic kidney disease in older diabetic adults. Kao YM[1], Chen JD. Ann Epidemiol. 2013 May;23(5):255-9

PURPOSE: To identify associations among body mass index (BMI), diabetes, and chronic kidney disease (CKD) in older adults in Taiwan.

METHODS:

This study enrolled 3334 participants aged 65 years and above who underwent an annual health screening at a medical center from January 2006 to December 2010. CKD was defined as an estimated glomerular filtration rate less than 60 mL/min/1.73 m(2). A multiple logistic regression analysis was used to determine associations among BMI, diabetes, and CKD.

RESULTS: The prevalence rate of CKD was 19.7% and 10.5% in diabetic and nondiabetic subjects, respectively. A multivariate model indicated that age, diabetes, hypertriglyceridemia, low levels of high-density lipoprotein cholesterol, and hyperuricemia were associated with an increased risk of CKD. Furthermore, there was an inverse association between BMI and CKD in older diabetic patients, with odds ratios of 3.71, 2.32, 2.12, and 1.31 in underweight, normal, overweight, and obese subjects, respectively, compared with nondiabetic subjects of normal weight.

CONCLUSIONS: There was an inverse association between BMI and CKD in older diabetic patients but no such association was found in nondiabetic older adults. More attention should be given to older underweight diabetic patients because they have a higher risk of CKD.

J Nephrol. 2012 May-Jun;25(3):384-93. doi: 10.5301/jn.5000062.

Body mass index has no effect on rate of progression of chronic kidney disease in subjects with type 2 diabetes mellitus.

Monsen A[*], Brown R, Hoefield R, Kalra PA, O Donoghue D, Middleton R, New D.M

BACKGROUND:

The incidences of obesity and chronic kidney disease (CKD) are reaching epidemic levels. Recently obesity has been associated with the development of CKD. However, it is unclear whether obesity is a risk factor for the progression of CKD. This study investigated the effect of raised body mass index (BMI, calculated as kg/m2) on the rate of CKD progression in a group of patients with CKD and type 2 diabetes mellitus.

METHODS:

The Chronic Renal Insufficiency Standards Implementation Study (CRISIS) is a large epidemiological study conducted in Manchester, UK. From the CRISIS database, we assessed the rate of progression of CKD in 229 adults who met the inclusion criteria. Baseline measurements such as BMI, estimated glomerular filtration rate (eGFR) and systolic and diastolic blood pressure were collected. eGFR measurements were obtained during follow-up to calculate the rate of eGFR change (ΔeGFR). Linear regression analysis and independent sample t-test were used in data analysis.

RESULTS:

After a mean follow-up period of 31 months, linear regression analysis showed no relationship between ΔeGFR and BMI. Furthermore, independent sample t-test comparing the obese (BMI =30) and nonobese (BMI <30) groups' ΔeGFR showed no statistical significance (p=0.572). Similar results were observed after stratification according to CKD stages 3, 4 and 5.

CONCLUSION: Raised BMI did not influence the rate of progression of chronic kidney disease in patients with type 2 diabetes mellitus.

Obesity Is a Risk Factor for CKD Risk Factors.

Obesity as an Independent Risk Factor for CKD.

BMI and ESRD

Case:

46yo women with ESRD secondary to chronic GN is placed on hemodialysis 3 times weekly

He has a BMI of 39 and his course on dialysis is characterized by barely adequate dialysis and difficult to control HTN and lipids

- How will obesity affect his mortality on dialysis?
- How will obesity affect his chances for transplantation?
- If he lost weight? Would it influence outcomes in either scenario?

Effects of overweight and obesity on the kidney

Hemodynamic

Structural

Pathologic

Chronic kidney disease

End-stage renal disease

1. ↑ Incidence and prevalence
2. Survival advantage in hemodialysis
3. ↑ Graft loss in kidney transplant recipients

Other

BMI and dialysis

- Low BMI (<22) is associated with increased risk of death, regardless of RRT modality

- Obesity (BMI >30) may confer a survival *advantage* with HD than with PD

Obesity and HD

The adverse effects of obesity :

- Difficulties in cannulation for vascular access
- Difficulties in making of arteriovenous fistula.
- Higher risk of atherosclerosis due to obesity,
- Higher risk of coronary and cerebral vascular disease.

The “obesity paradox”

There is a linear inverse relationship between mortality and BMI

(Reverse epidemiology)

Obesity paradox in other chronic diseases as myocardial infarction, heart failure, and COPD.

Published in final edited form as:
Am J Transplant. 2011 April ; 11(4): 725–736. doi:10.1111/j.1600-6143.2011.03468.x.

Associations of Body Mass Index and Weight Loss with Mortality in Transplant-Waitlisted Maintenance Hemodialysis Patients

Miklos Z Molnar, MD, PhD[1,2], Elani Streja, MPH[1,3], Csaba P Kovesdy, MD[4,5], Suphamai Bunnapradist, MD[6], Marcelo S Sampaio, MD[6], Jennie Jing, MS[1], Mahesh Krishnan, MD[6,7]

- Higher BMI in ESRD or dialysis patients is associated with a lower rather than higher mortality.
- Overweight or Obese patients on dialysis associated with a survival benefit, which observed in “healthier” as well as the “sicker” subgroups of patients.

GOOD WOMEN

They can bring balance to your life!

WHY

- Presentaion: Obese patients may be presenting earlier with less advanced disease compared to those with lower BMI.
- Cachexia associated with ESRD and other debilitating chronic diseases, may also contribute to higher mortality in lean compared to obese patients.
- Underweight may reflect a general poor health status associated with high mortality in these patients.
- Obese patients may be more hemodynamically stable.
- Protein-energy malnutrition less likely than lean.

BMI and Peritoneal Dialysis

Obese subjects :

less likely to initiate peritoneal dialysis,

less likely to undergo transplantation, and

more likely to switch to hemodialysis.

Snyder et al. Kidney International, Vol. 64 (2003), pp. 1838–1844

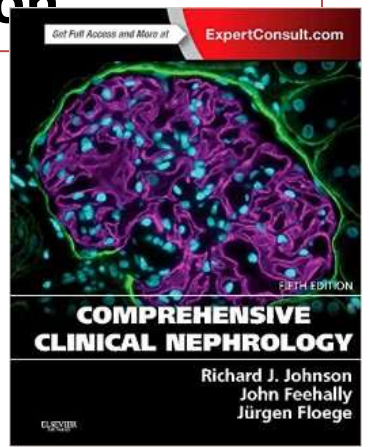
Contraindications to Peritoneal Dialysis

Severe Malnutrition

- Poor wound healing
- Leakage from the catheter tunnel
- Peritoneal protein losses during dialysis may exacerbate hypoalbuminemia

Morbid Obesity

- Difficult to place catheter through the abdominal wall
- Catheter leak, Exit site infections.
- inadequate solute clearance or ultrafiltration
- Peritonitis



Obesity and renal transplantation

Recipient Factors Affecting Graft and Patient Survival After Renal Transplantation

Children: Outcome worse <7 kg

Gender: Better outcomes from male donor to female recipient, presumably based on nephron mass.

Race: Worse outcomes compared with whites in United States for African Americans, but better for Asians. Variable effects in other countries but generally worse for indigenous races.

Anti-HLA antibodies: Worse with increasing sensitization because of previous blood transfusions, pregnancies, grafts.

Previous transplants: Worse outcomes with second and subsequent grafts.

Primary renal disease: Specific risks of recurrent disease by type of primary disease.

Comorbidities: Worse outcome with cardiovascular disease, chronic respiratory disease, diabetes mellitus, hepatitis B, or

Body mass index: Worse outcomes at extremes of BMI: below 20 and above 35

Transplantation-Related Factors

Surgical experience: Worse outcomes with inexperienced surgeons.

Graft and patient vascular anatomy: Worse outcomes with multiple arteries and veins.

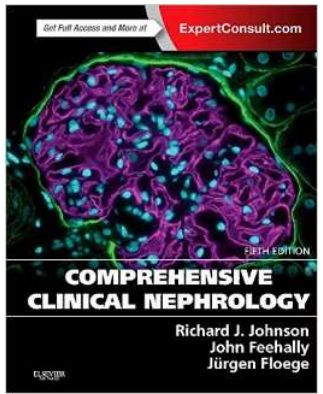
Warm ischemia time: Worse outcome with prolonged time.

Cold ischemia time: Worse outcome with prolonged time.

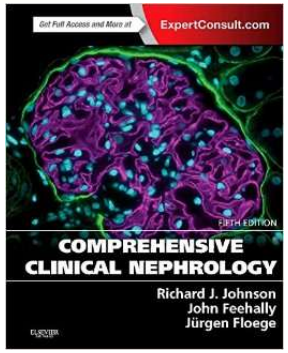
Transplant center experience and results: Most apparent center effects are related to patient and donor selection criteria rather than center expertise. However, some centers in some countries do produce worse outcomes based on multivariate analysis of all relevant factors, leaving center expertise the most likely remaining outcome variable.

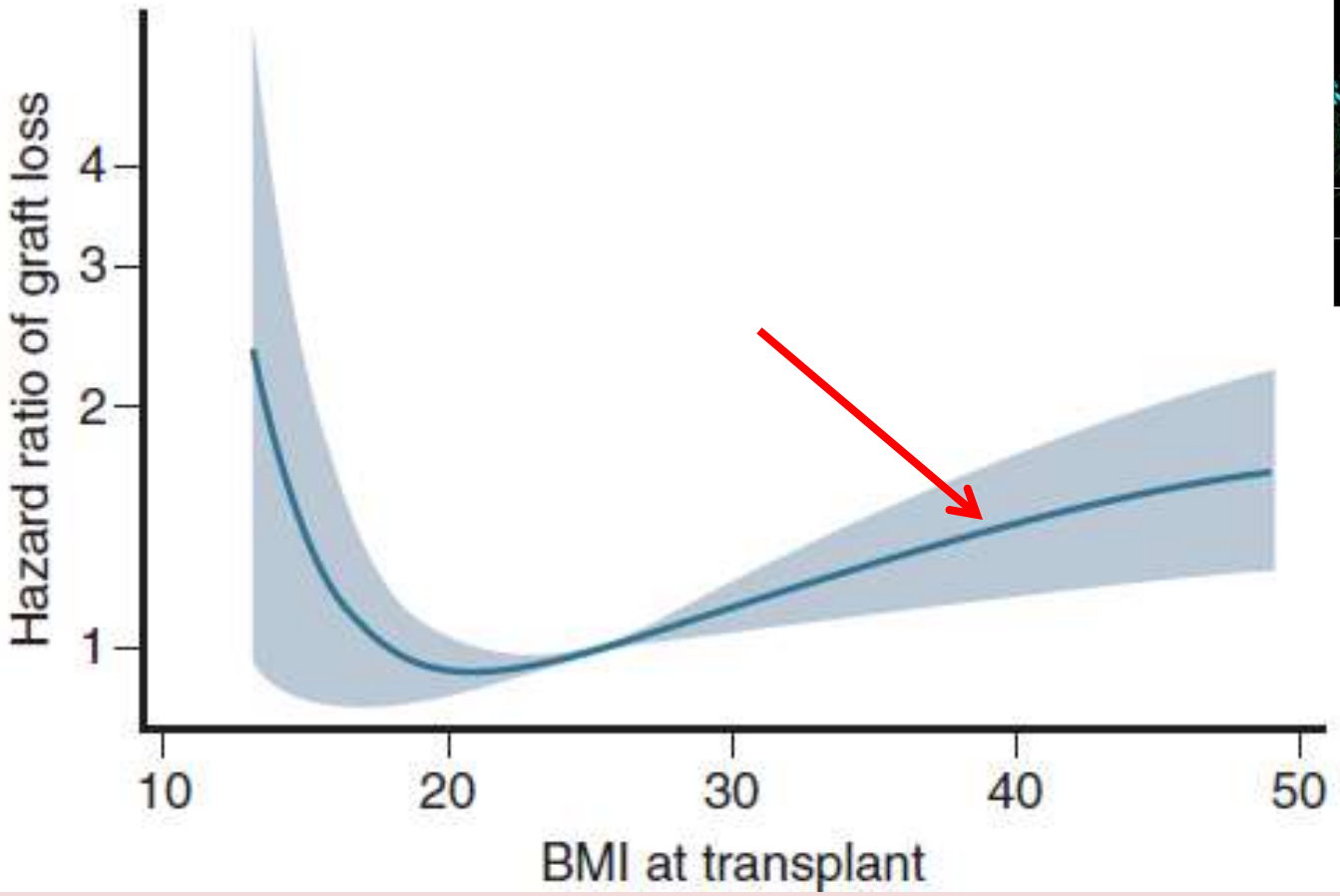
HLA matching: Worse outcome with poor matching.

Donor-specific antibodies: Easily detected high-titer anti-HLA class I and class II antibodies detected by cytotoxicity crossmatch tests are associated with hyperacute, accelerated, and acute rejection. Anti-HLA antibodies detected only by



Body Mass Index and Graft Loss





Univariate analysis of graft loss by body mass index in 5684 patients in ANZDATA from 1991 to 2004. *Blue line* shows hazard ratio of graft loss; *shaded area* shows confidence intervals.

Transplantation. 2002 Jan 15;73(1):70-4.

The impact of body mass index on renal transplant outcomes: a significant independent risk factor for graft failure and patient death.

Meier-Kriesche HU[1], Arndorfer JA, Kaplan B.

Abstract

INTRODUCTION:

Renal transplant recipients with elevated body mass index (BMI) have been shown to have inferior patient survival as compared to patients with lower BMI. However, previous studies could not establish a link between increased BMI and decreased death censored graft survival. Obesity in nontransplant patients has been associated with hypertension, hyperlipidemia, type II diabetes, proteinuria and glomerulopathy. Given this evidence it is possible that renal transplant recipients with an elevated BMI may have worse long term graft survival. To investigate this hypothesis we retrospectively analyzed 51,927 primary, adult renal transplants registered in the USRDS.

METHODS:

BMI at date of transplant was calculated for all patients using BMI=body weight (in kg)=.stature (height, in meters) squared. BMI values were further categorized into 11 categories: below 18, from 18 to 36 at 2 unit increments, and above 36 kg/m2. Primary study end points were graft and patient survival. Secondary study end points were death censored graft survival, chronic allograft failure, delayed graft function, and acute rejection (AR). Cox proportional hazard and logistic regression models investigated the link between categorized BMI and the study end points correcting for potential confounding variables.

RESULTS:

CONCLUSIONS:

BMI has a very strong association with outcomes after renal transplantation independent of most of the known risk factors for patient and graft survival. The extremes of very high and very low BMI before renal transplantation are important risk factors for patient and graft survival. It is important to note that elevated BMI was significantly associated with worse graft survival independent of patient survival. Whether prospective weight adjustment before renal transplantation can favorably affect posttransplant risk needs to be assessed by further studies.

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Effects of Body Mass Index at Transplant on Outcomes of Kidney Transplantation

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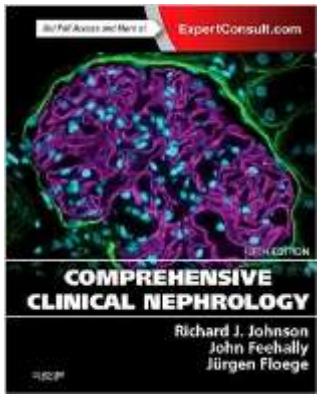
Backgound. While obesity increases postoperative complications and cardiovascular risks, its effects on long-term kidney transplant outcomes are less clear.

Methods. We used data from the Australian and New Zealand Dialysis and Transplant (ANZDATA) Registry to examine the relationships between body mass index (BMI, classified according to World Health Organization criteria) at transplant and transplant outcome. Patients starting renal replacement therapy from April 1991 and who received a single-organ, primary kidney transplant (when aged ≥16 years) from April 1991 to December 2004 were included, and followed up to death or December 2005. Survival outcomes adjusted for important covariates were analyzed using Cox models, and cause-specific failures by competing risks analysis. Analysis using BMI at various times posttransplant was also performed. Intermediate outcomes were delayed graft function (DGF) and any acute rejection at 6 months.

Conclusions. Obesity per se was not associated with poorer kidney transplant outcomes, although it was associated with factors that led to poorer graft and patient survival..

with greater odds for DGF (adjusted OR: 1.56 [1.23–1.97], *P*<0.001) and 6-month risk of acute

Overweight or obese patient is more likely to develop new-onset diabetes after transplantation, which can adversely affect graft and patient survival.



Conclusions

- Evidence suggests that obesity is a modifiable risk factor for kidney disases.
- The presence of proteinuria is actually the initial clinical evidence of involving kidney and obesity
- A significant percentage of CKD cases could be prevented by eliminating overweight and obesity.
- Obesity appears to be protective in some situations
- Obese patients do worse with transplant and weight loss pre-op is mandatory.


Thank
You!!